

Supporting Information

Brandl et al. 10.1073/pnas.1210366109

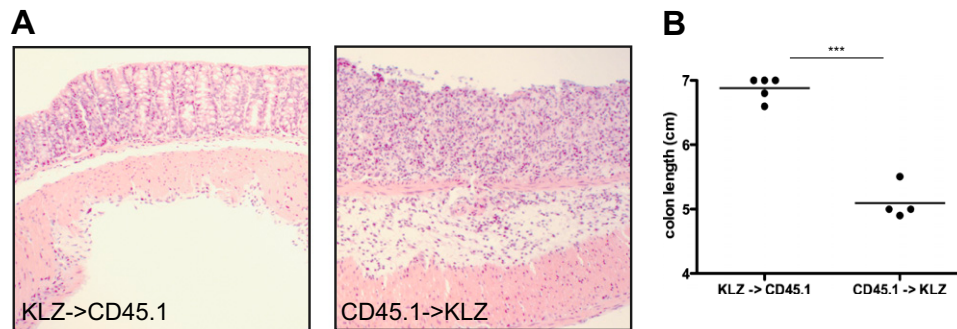


Fig. S1. Nonhematopoietic cells are responsible for the DSS colitis phenotype in *Klein-Zschocher* mice. (A) Representative photomicrographs (magnification 100 \times , H&E staining) of colons from different chimeric mice. (B) Colon length from different chimeric mice. $n = 4-5$ each group, unpaired t test, *** $P < 0.0001$. KLZ, *Klein-Zschocher*.

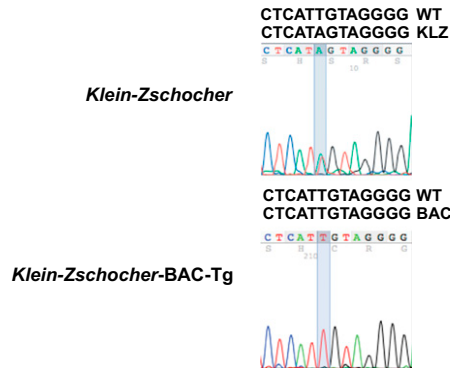


Fig. S2. Rescue of the *Klein-Zschocher* phenotype by BAC transgenesis. DNA sequence of *Yipf6* in the region of the *Klein-Zschocher* mutation in a heterozygous mouse (*Klein-Zschocher*), and in the BAC clone expressed as a transgene (*Klein-Zschocher-BAC-Tg*). The BAC clone contains the WT *Yipf6* sequence.

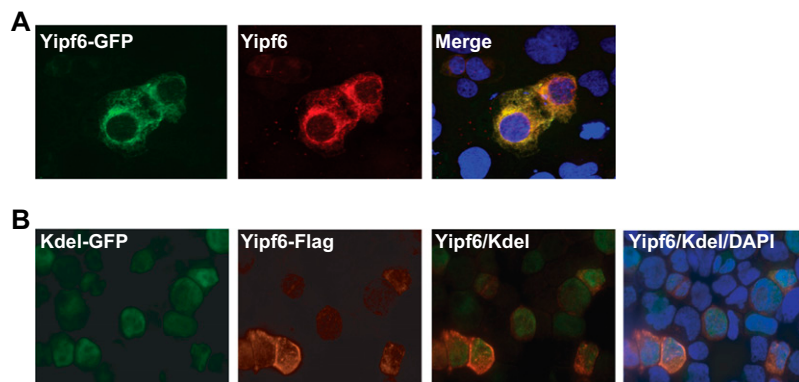


Fig. S3. *Yipf6* fails to colocalize with ER marker Kdel. (A) Polyclonal *Yipf6* antibody recognizes *Yipf6* protein. HEK293 were transfected with *Yipf6*-GFP and stained with a polyclonal *Yipf6* antibody and the colocalization of *Yipf6*-GFP and *Yipf6* was examined using confocal microscopy (magnification 600 \times). (B) Kdel-GFP protein was coexpressed together with Flag-*Yipf6* in HEK293 cells (magnification 400 \times).

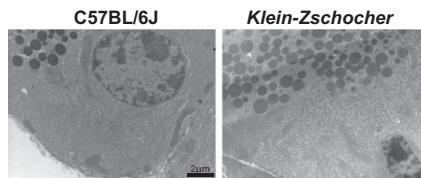


Fig. S4. Swollen ER in acinar cells from *Klein-Zschocher* mice. Electron microscopy of acinar cells from wild-type and *Klein-Zschocher* mice.